THE SYMPOTOMATOLOGY OF MOTION SICKNESS

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13. ABSTRACT

Motion sickness is maladaptation to a dynamic environment. The major symptoms are caused by inadequate and inappropriate vascular and circulatory responses, resulting mainly from inadequate perception (integration and analysis of the pertinent sensory data) of the dynamic environment and consequent misestimation of the nature and degree of the threat involved.

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The Symptomatology of Motion Sickness

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SUMMARY

Motion sickness is maladaptation to a dynamic environment. The major symptoms are caused by inadequate and inappropriate vascular and circulatory responses, resulting mainly from inadequate perception (integration and analysis of the pertinent sensory data) of the dynamic environment and consequent misestimation of the nature and degree of the threat involved.

INTRODUCTION

Motion sickness is essentially maladaptation to a novel inertial environment. The symptoms do not develop inevitably in the presence of motion, nor is motion inevitably present when the symptoms do develop (refs. 1 to 4). Motion-sickness symptoms occur only when there is a malfunction of the victim's orientation-perceiving mechanisms and his motion- and acceleration-compensating mechanisms. This is a complex system involving many subsystems. There are various ways in which it can fail or decompensate, depending on the specific nature of the load placed upon it and the individual variations in ability to handle the load.

The orienting system integrates and utilizes information from many sense organs, most notably the vestibular, the visual, and the tactile-proprioceptive-kinesthetic. There is much mutual interplay among these subsystems. The main systems utilizing the resulting integrated data are the visual, the musculoskeletal, and the cardiovascular. The system contributes to arousal as an important source of early warning of impending threat (danger of falling, being struck, etc.). It is also monitored for reliability. Both orientation information and an estimate of its reliability are available consciously. Either can warn of impending danger. The term "disorientation" refers to the condition in which perceived orientation is incorrect. The term "vertigo" (originally meaning a false sense of rotation) now is used commonly to refer to conscious awareness of a failing or inadequate perception of motion and accelerative forces. Conscious fear is relatively unimportant in motion sickness. Unconscious estimate of the threat is most significant. Adaptation to motion consists mainly of learning to perceive it correctly and to make proper adjustment of antigravity compensatory reflexes, etc., and learning to evaluate the threat properly so that inappropriate defensive preparatory steps are not taken.

Symptoms arise as the result of failure to make adequate compensatory adjustments, as the result of inappropriate or incorrect adjustments and preparations, and as a result of the additional information-processing load imposed by incorrect perceptual data processing and the effort to correct it. Similar symptoms can be produced by bringing about these same conditions in various ways, other than by subjecting the victim to the motions typically associated with motion sickness.

The novelty of the environment need not be so great as we might expect. Even return to that most familiar condition, 1 g vertical and zero angular acceleration and total zero velocity, can

produce symptoms in one who has been thoroughly adapted to some other, such as a ship, a rotating room, or even that environment produced by the disassociation of the normal relationship between the visual and the vestibular sensory inputs induced by wearing inverting or reversing spectacles (refs. 1 to 6). Such a condition could be called "still sickness" if we persist in attempting to name the disease by the characteristics of the environment that are contributory to its production.

Concepts of diseases lead lives of their own. They evolve. As we learn more about them, originally different diseases may come to be considered as but different manifestations of the same underlying disease process, or a disease may fragment into many as we learn to differentiate between basically different entities that had but superficial resemblance. As an illustration, I offer a disease from antiquity, the "boat disease." This is not a disease of boats but one caused by riding in boats. In accordance with medical tradition, it was named after the old Greek word for boat, "naus," hence nausea. This disease grew to include similar conditions even when they were not caused by riding in boats. Ultimately it lost its status as a disease and reverted to being merely a symptom found as part of many syndromes.

We went through the cycle again with seasickness which became travel sickness, which then fragmented into mountain sickness, trainsickness, carsickness, airsickness, etc. With the advent of laboratory interest in the condition, we acquired rotating chair sickness, Coriolis sickness, elevator sickness, swing sickness, and even Cinemascope sickness, 2–FH–2 Hoover simulator sickness, and still sickness. Motion sickness provides a pertinent and insightful name for the syndrome occurring under all of these circumstances. Even though motion is neither a necessary nor sufficient cause (and in the case of 2–FH–2 hover simulator sickness can, when properly applied, actually reduce symptom formation, preceding paper by Fred E. Guedry, Jr., "Conflicting Sensory Orientation Cues as a Factor in Motion Sickness"), motion does indicate the general area in which the victims are maladapted, whether with regard to their perception of the actual acceleration and motion environment or in the inadequacy of their compensatory physiological adjustments. "Maladaptation to inertial environment" more accurately (though awkwardly) designates the condition whose manifestations I am to discuss. For whether the subject is moving or not, whether his environs are moving or not, the inadequacy of his adaptation to the dynamic aspects of his environment is the one element that distinguishes the sick subject from the unaffected.

When examined closely, the borders separating one disease from another, one body malfunction from another, are not sharp. The blackout and unconsciousness that can result from high acceleration in the foot-to-head direction might not seem closely related to the usual symptoms of motion sickness. Yet the same symptoms would be felt every time we arose from a supine to an erect posture were it not for the cardiovascular compensatory adjustments that occur. Lesser derangements of these same adjustments can account for symptoms in many cases of motion sickness. The body is a complex system comprising many interrelated subsystems. Its malfunctions are better understood from this point of view than from attempts to sharply demarcate disease entities or to overemphasize the role of any one of the subsystems involved.

SYSTEMS INVOLVED IN MOTION SICKNESS

In motion sickness, the perceptual-sensory system dealing with inertia and motion, that part of the central nervous system that is alerted by and prepares for response against external threats, the cardiovascular system, and the neuromuscular system are involved. I offer a partial tracing of the vestibular signals as a rationale for dealing with a hypothetical central processor for dynamic inertial information rather than with detailed neural structures involved. The vestibular ganglia communicate with 14 specific neural structures having about 2 dozen mutual interconnections. These, in turn, communicate through approximately 120 identified channels to the next level consisting of 44 centers (ref. 7). Thus, going no farther than three steps from the

sense organ, and without even considering the visual, auditory, proprioceptive, and other inputs, the system becomes quite unwieldy.

Sense organs measure certain qualities of their environment and send signals to the central nervous system. To understand the subsequent chain of events, we must distinguish between the transmission of excitation and the transmission of messages. The distinction is particularly important in discussions of the vestibular system in which corresponding semicircular canals send the same message by shifting the intensity of their signals (repetition rate of their pulse outputs) in opposite directions. This is important in understanding how the reduced sensitivity found in Ménière's syndrome can lead to sensations of rotation, dizziness, and nausea. A reduced pulse output rate conveys the message of rotation as surely as does an increased output rate. A man who has recently lost all vestibular input on one side as a result of surgery is receiving from that side zero input pulses (i.e., zero excitation), but a message signifying very strong angular and linear acceleration. Analysis of the production of symptoms in motion sickness is analysis of changes occurring in the body as the result of all incoming messages concerning orientation, acceleration, and motion (or failing to occur in appropriate response to this information).

Sensory organs function normaliy in motion sickness. At least there is no evidence that they are not functioning normally, and indeed it is extremely difficult if not impossible to elicit motion-sickness symptoms in subjects lacking functional inertia-sensitive sense organs. The central processor is usually not working correctly. Perception of acceleration and motion is usually (though not necessarily always) incorrect. Symptoms arising directly from this malfunction are minimal, consisting mainly of such illusions as a tilting horizon. Failure of correct central interpretation of acceleration and motion data participates in the generation of major symptoms mainly through the inappropriate or inadequate adjustments of the other subsystems that require the information for their proper functioning and through the elicitation of attempts to correct the central perceptual processor.

The cardiovascular system is a major participant in symptom generation, first through failure to compensate adequately for acceleration loads placed directly on it, and, second, by its inability to handle the demands placed on it by the circulatory requirements of muscles inappropriately preparing for emergency action. The neuromuscular system participates by its inordinate demands on the circulatory system and manifests the inadequate central integration of inertial data by ataxia, tenseness, and fatigue. The central arousal system (reticular formation?) participates by triggering several alarm responses, including muscle hyperemia and, more appropriately, the reorganization of inertial perception. Many of the symptoms and other observable conditions of motion sickness are overdetermined. There are several causal chains leading to the same effect. Some of the changes observed are part of the problem, some part of the solution, and some are both. A change compensating for one disturbance may aggravate another.

Inadequate cerebral circulation is an old theory of motion sickness. It still has much to recommend it. There can be no doubt that nausea often accompanies decreasing blood pressure and falling cardiac output from any cause. Whether or not this nausea is secondary to embarrassed cerebral circulation is less certain. Two items are of cerebral significance in motion sickness. One is the known increased metabolic demands of the brain in arousal states (ref. 8), and the other is the high correlation between susceptibility to motion sickness and unusual lability of cerebral circulation (at least as reflected by central retinal artery changes) under conditions of minor longitudinal g-changes on a horizontal swing (ref. 9). There is some experimental evidence that cerebral circulation, or at least the quantity of blood in the head, decreases with vestibular stimulation (ref. 10). Decreased spontaneity, increased depression, and headache are mild indications of impaired cerebral function.

Compensation for longitudinal acceleration is about the simplest adjustment the vascular system must make. Yet it is of considerable magnitude. For even so simple a change as shifting from the supine to the erect posture, the hydrostatic pressure difference introduced between

head and foot is greater than the pressure difference that the heart maintains between its input and its output. To remain adequate, this adjustment requires practice and training. Lightheadedness accompanies first arising after several days in bed. Symptoms may be caused simply by vascular compensatory inadequacy in spite of adequate correction data supplied by the central integrater of inertial data. The afternausea following various types of motion sickness experiments is aggravated by standing, relieved by sitting or reclining. One of the mildest inputs, from the point of view of sensory stimulation, is capable of producing nausea in a few minutes. This is simple rotation in a vertical plane at a frequency of about 15 rpm while in the seated position (ref. 11). Elevator experiments have shown that g-changes at about this frequency are most effective in nausea production, though more time is required (ref. 12). Some have attributed this frequency sensitivity of the nausea-producing mechanism to an undiscovered resonance in the sense organs. I feel that this is a highly unlikely explanation, and far more significant is the fact that this is approximately the natural slosh frequency of the blood in the vascular system. Near this frequency the blood undergoes the greatest displacement, and the greatest compensatory shift in vessel tone is required.

In zero-g experiments, in which zero g alternates with increased g, the periods of increased g seem most responsible for the symptoms. In one series, subjects either became sick during the preweightless acceleration or they did not become sick at all (ref. 13). In another series in which 51 percent of the subjects vomited at one time or another, no one experienced nausea while prone during the accelerations. Two vomited in this position, but this occurred within 20 seconds of starting the maneuver and with no nausea (ref. 14). It appears that for production of nausea under these circumstances, increased loads on the cardiovascular system are of greatest importance. I participated in two such runs. Allowed to lie down during increased g and to float free during zero g, I experienced no symptoms. On the later run, confined to a seat, I became nauseated and experienced afternausea hours later while standing.

The cardiovascular changes considered up to this point could occur in the absence of any disorientation or errors in data processing relevant to motion and inertia. Errors in processing inertial information can only make matters worse. Even in that excellent angular overstimulation situation, Coriolis stimulation, linear-acceleration messages cannot be ignored. Angular acceleration about any axis not parallel to the direction of linear acceleration implies a change of direction of linear g. Central-nervous-system determination of direction of linear g (vertical) is a complex thing involving several separate inputs. In general, the short-term information is derived from semicircular-canal inputs, and the long-term information depends on integration of inputs from the otolith and other organs. With a time constant of only a few seconds for blood displacement caused by linear g, compensation, to be adequate, must utilize some of the implied g-change information based on semicircular-canal inputs. Experimental subjects report that they experience changes in linear g. They describe this as being in a climbing or diving spiral. Objective measurements on the same subjects show actual and different physical responses, depending on whether a climb or a dive is being experienced. Some subjects showed slowed and deepened respiration during the dive," and breath holding during the "climb."

Neuromuscular Factors

To keep the cause-effect steps in proper sequence, I should like to discuss the neuromuscular changes before returning to more serious cardiovascular problems that occur secondarily to the muscle changes. The simplest neuromuscular effects are directly caused by improper central-nervous-system integration and interpretation of sensory messages dealing with acceleration and motion. The problem is made more difficult by the need to predict the inertial environment in order that intended movements and dynamic postural reflexes may be properly compensated in time to achieve their objectives. Correcting muscle tension after an arm or leg has been deflected by an improperly predicted force is not a satisfactory solution. We are

usually unaware of these unconscious compensatory contributions to our motions, though we may experience them as a heaviness on first emerging after some time in the water, or as a lightness when first dropping a heavy load. Without the correct sensing and utilization of acceleration data, we experience ataxia and clumsiness. This is somewhat inconvenient and potentially dangerous, but in most motion-sickness situations greater harm is caused by the body's reaction to what is only potentially dangerous. The part of the alarm reaction that prepares the muscles for anticipated strong action in the face of this implied environmental threat contributes greatly to severe motion sickness.

Immediate reflex response to acceleration and motion can contribute to tenseness and subsequent fatigue. Benson has found that the gastrocnemius and soleus reflexes in man are increased by angular acceleration (ref. 15). There is no compensatory decrease in the opposite leg, merely a lesser increase. This increase in 16 subjects was from 73 percent to 136 percent, always greater in the trailing leg and linearly related to the table velocity before deceleration over a range from 20° to 110°/sec. The reaction required the presence of a functioning labyrinth and was apparently mediated by the gamma efferent system, rather than directly via the alpha motoneurons. The monosynaptic reflexes were unaltered. There is indirect evidence for the involvement of the extrapyramidal system in motion sickness. Most antihistamine drugs effective in motion sickness are also effective in Parkinsonism, and a surprisingly large number of drugs primarily identified as effective in Parkinsonism have proven effective in motion sickness (ref. 16). The involvement of the system for dynamic postural reflexes seems evident in both cases.

The greatest contribution of the neuromuscular system to the generation of motion-sickness symptoms is probably through the part of the alarm reaction that prepares the muscles for vigorous activity. Increase in blood sugar appears to be part of the solution rather than a cause of symptoms (ref. 17 and Fields, Meakins, and MacEachern, cited in ref. 3, p. 1816). Reducing blood sugar by giving insulin increases the symptoms. Other indications of alarm response are increased blood and urine levels of catecholamines and 17-hydroxycorticosteroids (ref. 18). These changes also probably do not contribute to symptoms, but rather to their suppression. Sympatholytic drugs increase the symptom formation while sympathomimetic drugs tend to reduce symptoms (ref. 19). One phenothiazine derivative that might have been expected to be helpful turned out not to be, but it differed from the effective drugs in being sympatholytic. The most significant preparatory move by far, however, is the diversion of circulating blood to the muscles.

It has been shown that muscle volume increases in subjects who are becoming motion sick (ref. 10). This volume shift in preparation for exercise is quite sensitive to central neural control. Weber showed an increase in muscle mass as a response to merely thinking about exercise (refs. 20 and 21). Apparently, actually using the muscles tends to counteract some of the bad effects. Physical work and going about one's business tend to reduce the symptoms (ref. 17). Circulatory compensation for the mass of blood diverted to muscles is difficult. Skin blanching can compensate for only part of it (ref. 10). Other organs, mainly the intra-abdominal viscera, must also lose some of their blood supply.

Cardiovascular Factors

Having described the manner in which muscle preparation for vigorous action can place additional loads on the cardiovascular system, I should like to examine the evidence that such a load does actually occur and that failure to adjust to it correlates highly with development of symptoms.

The most severe motion-sickness symptoms seem to be caused by a decrease in circulating blood volume. In motion-susceptible individuals, pulse rate decreases, systolic pressure and minute volume decrease (refs. 10, 22, and 23). The common features are indicative of a precollapse state. This is indicated primarily by the sharp drop in systolic pressure and minute volumes in spite of increasing peripheral resistance of the arterial system. The body's own estimate of inadequate circulation is indicated by

increased output of antidiuretic hormone (ref. 24).

Stimulation of the VIIIth nerve, whether caloric, galvanic, or by angular acceleration, causes a fall in blood pressure (ref. 25). This fall in blood pressure can be blocked by cutting the vagus. Stimulation of the peripheral end of the cut vagus produces a similar fall in blood pressure. Lergigan, a phenothiazine derivative, can block the blood-pressure fall produced in either of these ways (ref. 19). It is an effective anti-motion-sickness drug. On the other hand, another phenothiazine derivative, chlorpromazine, a potent and specific antiemetic, is ineffective in motion sickness (ref. 26). This is probably because it lowers blood pressure and can, in large doses, cause vascular syncope. Measures that help combat circulatory collapse reduce motion-sickness symptoms. These are tight abdominal belts (ref. 27), anti-*g*-suits, intravenous dextran solution, and adrenergic drugs (ref. 19; and Enquist, cited in ref. 19). Other conditions that reduce cardiac output, such as cardiac tamponade or sudden congestive heart failure, tend to produce nausea.

Reduced cardiac output can produce symptoms in two ways: those secondary to attempts at compensation for the condition and those resulting from inadequate compensation. Blanching of skin and abdominal viscera appear to be compensatory. There is little evidence of increased acid production in motion sickness, but blanching would reduce mucosal resistance to that acid which is present. As high as 50 percent of chronic seasick subjects show anatomical changes in the gastric mucosa as a result of such irritation (ref. 28). Sweating, which commonly accompanies the blanching of the skin, could be compensatory for the skin's reduced effectiveness as a cooling organ. Sweating could also be part of the preparation for vigorous muscle action in anticipation of increased heat production. There are no reports on the actual body temperature of motion-sick subjects, though their common desire for cooler surroundings would indicate the impairment of heat-loss mechanisms.

It is not known for certain how decreased cardiac output and blood pressure produce nausea. There are several reasons to believe that it involves embarrassment of cerebral circulation and metabolism. Decreased oxygen tension seems responsible for the nausea of mountain sickness. There is a higher incidence of motion sickness in unpressurized aircraft flying at higher altitudes. Vestibular stimulation has promptly reduced the volume of blood in the brains of dogs and monkeys (ref. 8). Measures that tend to improve cerebral circulation can reduce symptoms without necessarily improving the general level of cardiac output. Lowering the head, for example, in a maneuver similar to that employed for the prevention of vascular syncope, can relieve the nausea (ref. 23). Nausea is less on sitting than on standing, and still less on reclining. Subjects with poor regulation of cerebral circulation are more susceptible to motion sickness (ref. 9). Retching or vomiting does, at least momentarily, raise the intracranial blood pressure. This is the only compensatory value observable for such a reflex, other than removing irritants from the gastrointestinal tract. It is notable that nausea accompanies cerebral circulatory embarrassment produced by various other causes.

CONCLUSION

The human body is a very complex system. In attempting to understand some malfunction we should not delve so deeply into one subsystem that we lose sight of the others. Previously, seeking the cause of motion sickness, I have denigrated the role of the sensory systems and emphasized that of the perceptual or central integrative system, but of course without sensory input there is nothing to perceive or integrate (ref. 29). In preparing the present paper on symptomatology, I was impressed by the role of the extrapyramidal and vascular systems. Both Parkinsonism and increased susceptibility to motion sickness have been reported as after-effects of viral encephalitis. A large number of the same drugs are useful in both conditions. Dynamic postural reflexes are increased under conditions of acceleration. Muscle volume also increases.

The major symptoms seem to be caused by cardiovascular inadequacy, secondary to diversion of circulating blood to the muscles in response to a threatened need for vigorous

muscular action on the basis of inadequately perceived inertial and dynamic environment. The problem is aggravated in people with poor cerebral circulatory control. Vagotonic subjects are more susceptible. Cholinergic drugs aggravate and anticholinergic drugs ameliorate the symptoms. Two of the most effective anti-motion-sickness drugs, scopolamine and amphetamine, have little else in common except perhaps their usefulness in Parkinsonism. One blocks the vagus-mediated fall in blood pressure and cardiac output. The other increases blood pressure and cardiac output. These effects are additive against motion sickness.

A disease that began in the stomach has moved through the ears, the brain, the musculature, the visual, the circulatory, the endocrine, the thermoregulatory, the urinary, and back to the upper intestine (increased serotonin content). What have we overlooked?

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